CHAPTER 19

Trauma

Eyelid trauma

Haematoma

A haematoma (black eye) is the most common result of blunt injury to the eyelid or forehead and is generally innocuous. It is, however, very important to exclude the following more serious conditions:

1. **Trauma to the globe or orbit**. It is easier to examine the integrity of the globe before the lids become oedematous (Fig. 19.1).
2. **Orbital roof fracture**, if the black eye is associated with a subconjunctival haemorrhage without a visible posterior limit (Fig. 19.2).
3. **Basal skull fracture**, which may give rise to characteristic bilateral ring haematomas ('panda eyes') (Fig. 19.3).



Fig. 19.1
Severe periocular haematoma



Fig. 19.2
Periocular haematoma and subconjunctival haemorrhage



Fig. 19.3
'Panda eyes'

Laceration

The presence of a lid laceration, however insignificant, mandates careful exploration of the wound and examination of the globe. Any lid defect should be repaired by direct horizontal closure whenever possible, even if under tension, since this affords the best functional and cosmetic results (Thaller principle).

1. **Superficial** lacerations parallel to the lid margin without gaping can be sutured with 6-0 black silk. The sutures are removed after 5 days.
2. **Lid margin** lacerations invariably gape (Fig. 19.4a) and must therefore be very carefully sutured with perfect alignment to prevent notching.
   a. Evaluation for possible tissue loss.
   b. Conservative trimming of any irregular edges or grossly contaminated tissue (Fig. 19.4b).
   c. Initial eyelid margin realignment with a 6-0 black silk suture placed through the meibomian gland orifices. The bite should extend 2 mm on each side of the wound edge and be about 1 mm in depth (Fig. 19.5a).
   d. The tarsal plate is closed (Fig. 19.5b) using partial thickness long-acting absorbable sutures such as 6-0 polyglycolic acid (Dexon).

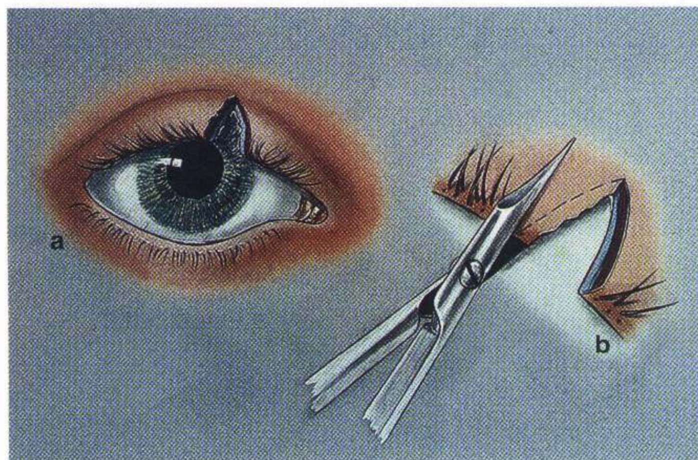


Fig. 19.4
Technique of suturing a marginal lid laceration (*see text*)
(Courtesy of Wilmer Institute)

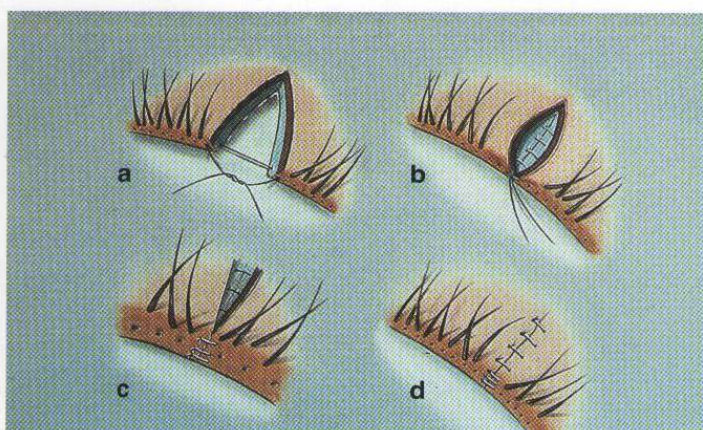


Fig. 19.5
Technique of suturing a marginal lid laceration (*see text*)
(Courtesy of Wilmer Institute)

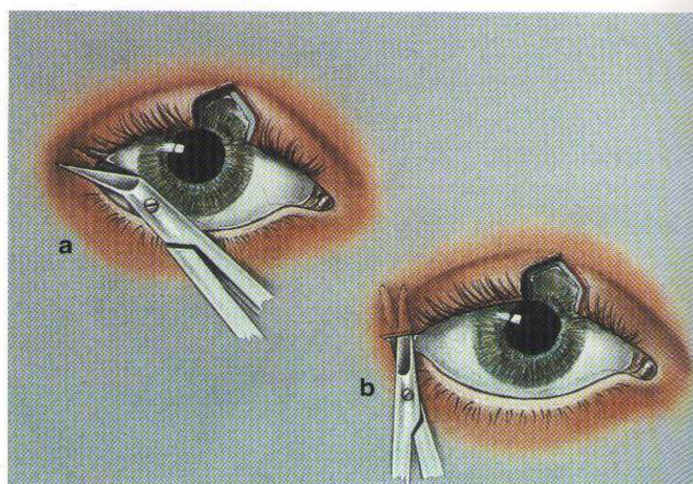


Fig. 19.7
Cantholysis (Courtesy of Wilmer Institute)

e. An additional lash line 6-0 silk suture is placed to achieve precise alignment of the tarsal margin and lashes (Fig. 19.5c).
f. The skin is closed with interrupted 6-0 black silk sutures (Fig. 19.5d).
g. The skin sutures are removed after 7–10 days.

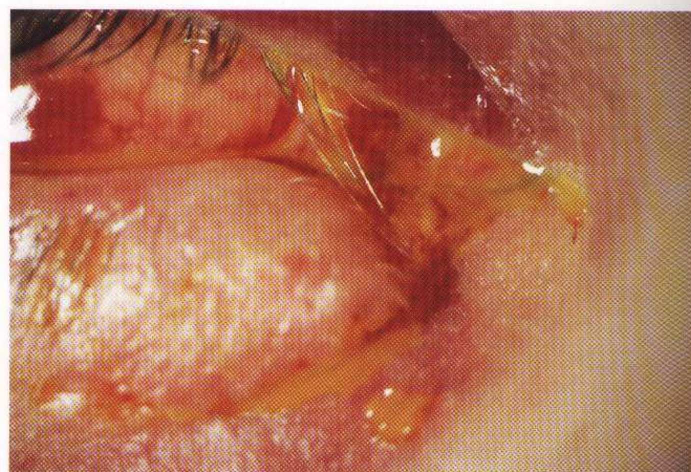


Fig. 19.8
Laceration involving the inferior canaliculus



Fig. 19.6
(a) Laceration of the lower lid; **(b)** after suturing

Figure 19.6 shows the pre- and postoperative appearances.

3. **Lacerations with tissue loss** just sufficient to prevent direct primary closure can usually be managed by performing a lateral cantholysis (Fig. 19.7) in order to increase lateral eyelid mobility.
4. **Lacerations with extensive tissue loss** may require major reconstructive procedures such as used following lid resection for malignant tumours (*see* Chapter 1).
5. **Canalicular lacerations** (Fig. 19.8) should be repaired within 24 hours.
   a. The laceration is bridged by silicone tubing, which is threaded down the lacrimal system and tied in the nose (Figs 19.9, 19.10 and 19.11 illustrate the principles of lacrimal intubation).
   b. The laceration is sutured.
   c. The tubing is left in situ for 3–6 months.

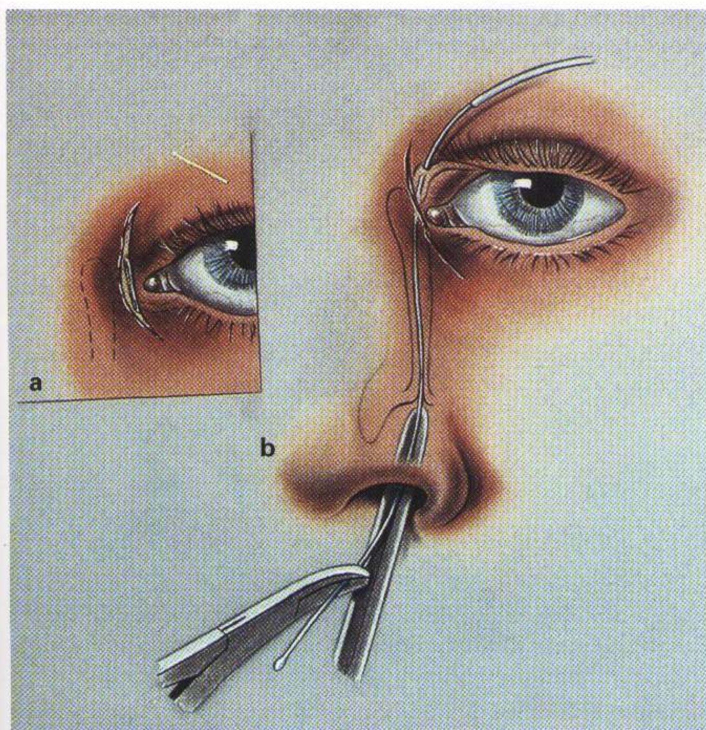


Fig. 19.9
Intubation of the lacrimal system (*see text*) (Courtesy of Wilmer Institute)

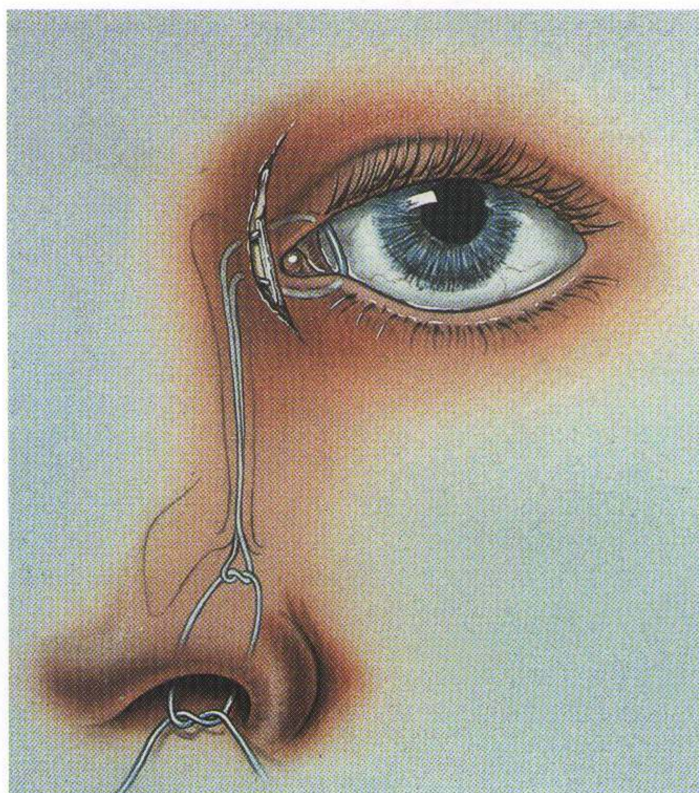


Fig. 19.10
Intubation of the lacrimal system (*see text*) (Courtesy of Wilmer Institute)

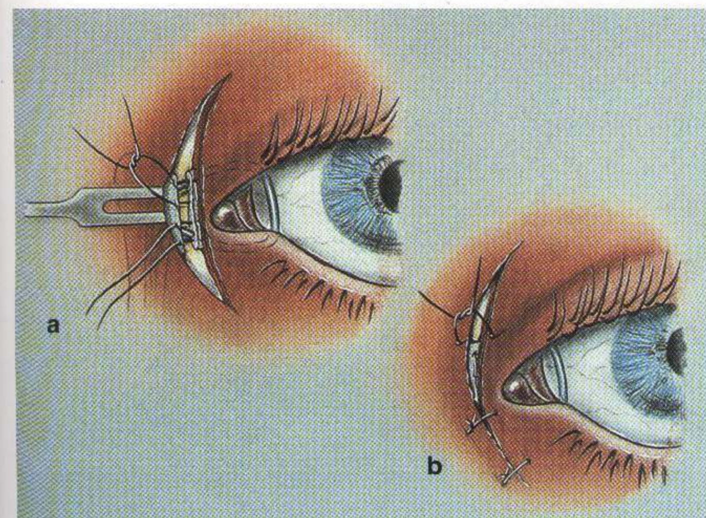


Fig. 19.11
Intubation of the lacrimal system (*see text*) (Courtesy of Wilmer Institute)

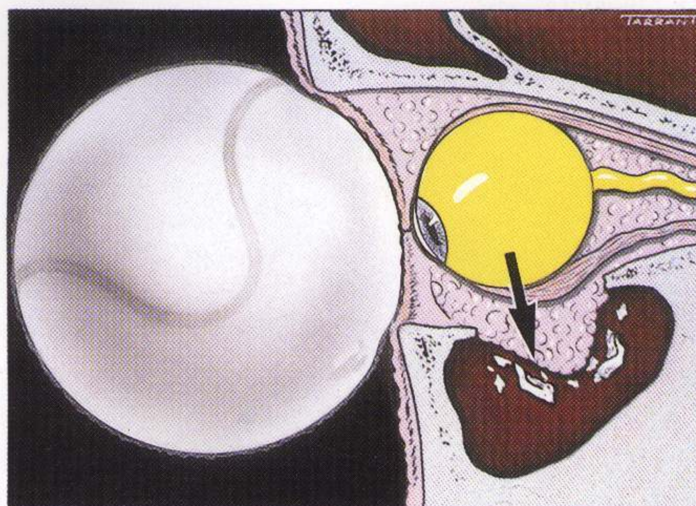


Fig. 19.12
Mechanism of an orbital floor blow-out fracture

Orbital fractures

Blow-out orbital floor fracture

A 'pure' blow-out fracture of the orbit does not involve the orbital rim whereas an 'impure' fracture involves the orbital rim and adjacent facial bones. A blow-out fracture of the orbital floor is typically caused by a sudden increase in the orbital pressure by a striking object which is greater than 5 cm in diameter, such as a fist or tennis ball (Fig. 19.12). Since the bones of the lateral wall and the roof are usually able to withstand such trauma, the fracture most frequently involves the floor of the orbit along the thin bone covering the infraorbital canal. Occasionally, the medial orbital wall may also be fractured. Clinical features vary with the severity of trauma and the time interval between injury and examination.

Signs

1. **Periocular signs** include variable ecchymosis, oedema and subcutaneous emphysema (Fig. 19.13a).
2. **Infraorbital nerve anaesthesia** involving the lower lid, cheek, side of nose, upper lip, upper teeth and gums is very common because a blow-out fracture frequently involves the infraorbital canal.
3. **Diplopia** may be caused by one of the following mechanisms:
   - Haemorrhage and oedema of the orbit may cause the septa, which connect the inferior rectus and inferior oblique muscles to the periorbita, to become taut and thus restrict movements of the globe. Ocular motility usually improves as haemorrhage and oedema resolve.
   - Mechanical entrapment within the fracture of the inferior rectus or inferior oblique muscle, or adjacent connective tissue and fat. Diplopia typically occurs in both upgaze (Fig. 19.13b) and downgaze (double diplopia). In these cases the forced duction test and the differential intraocular pressure tests are positive. Diplopia may subsequently improve if mainly due to entrapment of connective tissue and fat, but usually persists if there is significant involvement of the muscles themselves.
   - Direct injury to an extraocular muscle is associated with a negative forced duction test. The muscle fibres usually regenerate and normal function returns within about 2 months.
4. **Enophthalmos** (Fig. 19.13c) may be present if the fracture is severe, although it tends to manifest after a few days, as the initial oedema resolves. In the absence of surgical intervention, enophthalmos may continue to increase for about 6 months as post-traumatic orbital degeneration and fibrosis develop.
5. **Ocular damage** (e.g. hyphaema, angle recession, retinal dialysis), although uncommon, should be excluded by careful slit-lamp and fundus examination.

Investigations

1. **CT** with coronal sections (Fig. 19.14) is particularly useful in evaluating the extent of the fracture, as well as determining the nature of maxillary antral soft-tissue densities which may represent prolapsed orbital fat, extraocular muscles, haematoma or unrelated antral polyps.
2. **Hess test** (Fig. 19.15) is useful in assessing and monitoring the progression of diplopia.
3. **Field of binocular vision** can be assessed on the Lister or Goldmann perimeter.







Fig. 19.13
Right orbital floor blow-out fracture. **(a)** Mild bruising, superficial laceration and dilated pupil following fundus examination; **(b)** restricted elevation; **(c)** mild enophthalmos

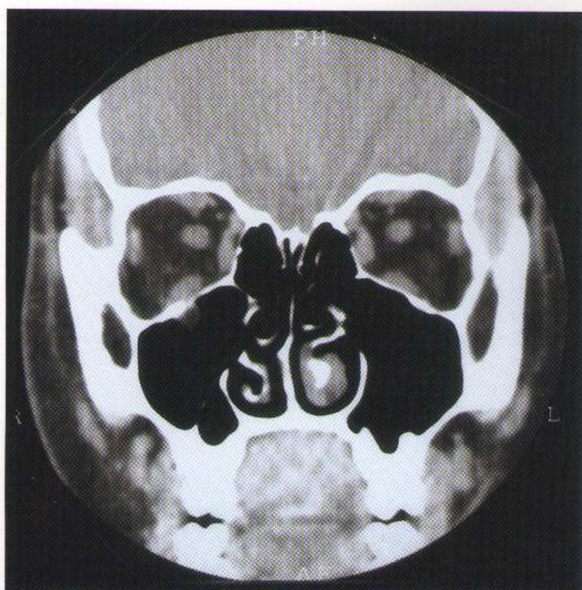
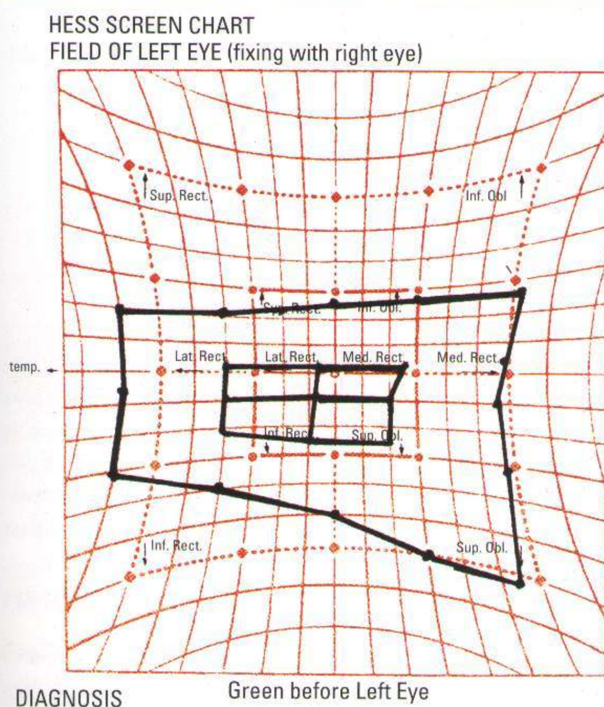


Fig. 19.14
Coronal CT scan of a right orbital floor blow-out fracture showing the 'tear drop' sign

Treatment

1. **Initial treatment** is conservative with antibiotics if the fracture involves the maxillary sinus. The patient should also be instructed not to blow the nose.
2. **Subsequent treatment** is aimed at prevention of permanent vertical diplopia and/or cosmetically unacceptable enophthalmos. The three factors that determine the risk of these late complications are fracture size, herniation of orbital contents into the maxillary sinus and muscle entrapment. Although there may be some overlap, most fractures fall into one of the following categories:
   - Small cracks unassociated with herniation do not require treatment as the risk of permanent complications is small.
   - Fractures involving less than half the orbital floor, with little or no herniation, and improving diplopia also do not require treatment unless more than 2 mm of enophthalmos develops.
   - Fractures involving half or more of the orbital floor with entrapment of orbital contents and persistent diplopia in the primary position should be repaired within 2 weeks. If surgery is delayed, the results are less satisfactory because secondary fibrotic changes develop in the orbit.
3. **Technique of surgical repair**
   a. A transconjunctival or subciliary incision is made (Fig. 19.16a).
   b. The periosteum is elevated from the floor of the orbit and all entrapped orbital contents are removed from the antrum (Fig. 19.16b).
   c. The defect in the floor is repaired by using synthetic material such as Supramid, silicone or Teflon (Fig. 19.16c).



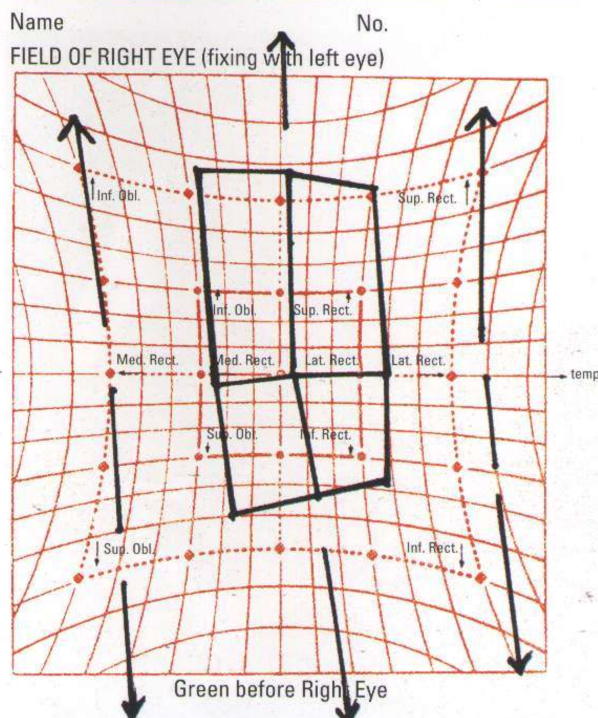


Fig. 19.15
Hess chart of a left orbital floor blow-out fracture showing restriction of left upgaze (superior rectus and inferior oblique) and restriction on downgaze (inferior rectus). There is also considerable secondary overaction of the right eye

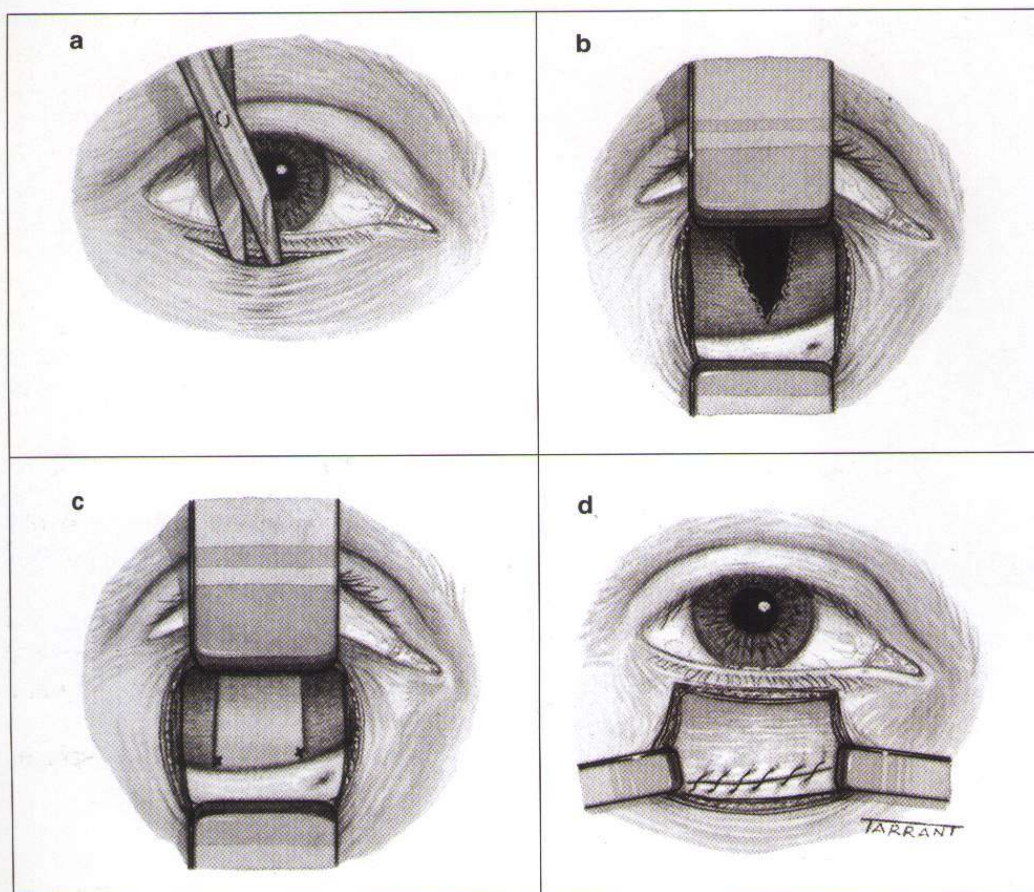


Fig. 19.16
Technique of repair of an orbital floor blow-out fracture (*see text*)

d. The periosteum is sutured (Fig. 19.16d).
Figure 19.17 is a coronal CT scan showing the post-operative appearance following repair of a right blow-out fracture with a plastic implant.



Fig. 19.17
Coronal CT scan following repair of a right orbital floor blow-out fracture with synthetic material

Blow-out medial wall fracture

Most medial wall orbital fractures are associated with floor fractures (Fig. 19.18). Isolated fractures are less common.

1. Signs
- Periorbital subcutaneous emphysema (Fig. 19.19), which typically develops when the patient blows the nose. Because of the possibility of forcing infected sinus contents into the orbit, blowing of the nose should be discouraged.
- Defective ocular motility involving adduction and abduction (Fig. 19.20), if the medial rectus muscle is entrapped in the fracture.

2. Treatment involves release of the entrapped tissue and repair of the bony defect.

Roof fracture

Roof fractures are rarely encountered by ophthalmologists. Isolated fractures, caused by minor trauma such as falling on a sharp object or a blow to the brow or forehead, are most common in young children. Complicated fractures, caused by major trauma with associated displacement of the orbital rim

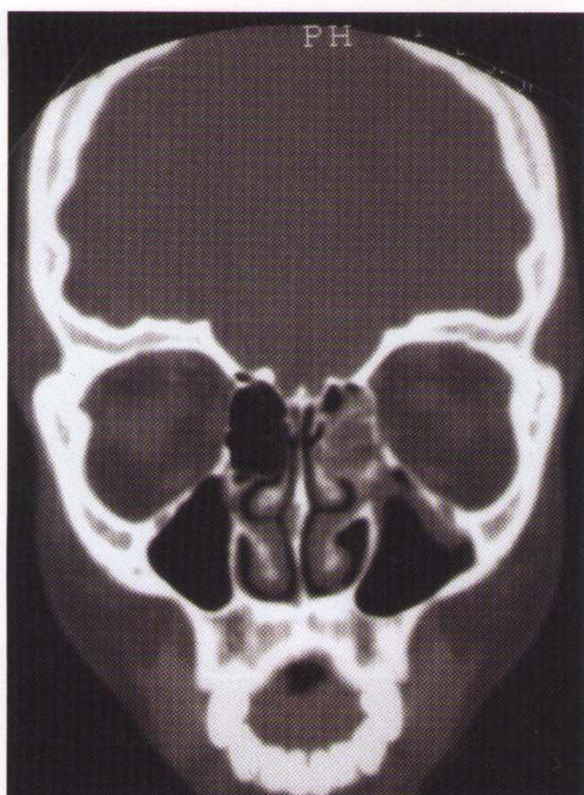


Fig. 19.18
Coronal CT scan showing blow-out fractures of the left medial orbital wall and floor



Fig. 19.19
Subcutaneous emphysema in a patient with a medial orbital wall blow-out fracture

or significant disturbance of other craniofacial bones, most commonly affect adults.

1. **Presentation** is with a haematoma of the upper eyelid and periocular ecchymosis which develop after a few hours and may later spread to the opposite side (*see* Fig. 19.3).
2. **Signs**
   - Inferior or axial displacement of the globe.
   - Large fractures may be associated with pulsation of the globe unassociated with a bruit, due to transmission of CSF pulsation, best detected on applanation tonometry, if possible.



Fig. 19.20
Limitation of left abduction due to entrapment of the left medial rectus muscle in a medial orbital wall blow-out fracture

3. **Treatment**
   - Small fractures may not require treatment but it is important to observe the patient for the possibility of a CSF leak which may lead to meningitis.
   - Sizeable bony defects with downwardly displaced fragments usually require reconstructive surgery.

Lateral wall fracture

Acute lateral wall fractures (Fig. 19.21) are rarely encountered by ophthalmologists. Because the lateral wall of the orbit is more solid than the other walls, a fracture is usually associated with extensive facial damage.

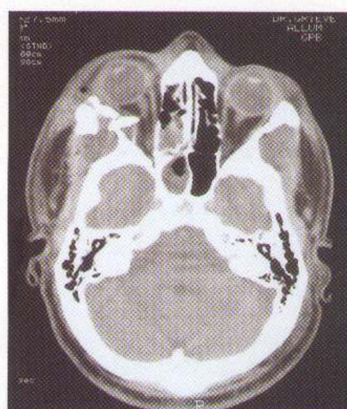


Fig. 19.21
Axial CT scan showing a right lateral wall fracture with bony fragments impinging on the optic nerve

Trauma to the globe

Introduction

Definitions

1. **Closed injury** is commonly due to blunt trauma. The corneoscleral wall of the globe is intact; however, intraocular damage may be present.

2. **Open injury** involves a full-thickness wound of the corneoscleral wall.
3. **A contusion** is a closed injury resulting from blunt trauma. Damage may occur at the site of impact or at a distant site.
4. **A rupture** is a full-thickness wound, caused by blunt trauma. The globe gives way at its weakest point, which may not be at the site of impact.
5. **A laceration** is a full-thickness wound caused by a sharp object at the site of impact.
6. **A lamellar laceration** is a partial-thickness wound caused by a sharp object.
7. **Penetration** is a single full-thickness wound, usually caused by a sharp object, without an exit wound. Such a wound may be associated with intraocular retention of a foreign body.
8. **Perforation** consists of two full-thickness wounds, one entry and one exit, usually caused by a missile.

General principles of management

1. **Initial assessment** should be performed in the following order:
   - Determination of the nature and extent of any life-threatening problems.
   - History of the injury, including the circumstances, timing and likely object.
   - Thorough examination of both eyes and orbits.
2. **Special investigations**
   a. ***Plain radiographs*** may be taken when a foreign body is suspected (Fig. 19.22).
   b. ***CT*** is superior to plain radiography in the detection and localization of intraocular foreign bodies (Fig. 19.23). It is also of value in determining the integrity of intracranial, facial and intraocular structures.

> ***NB:*** MRI should never be performed if a metallic foreign body is suspected.

   c. ***Ultrasonography*** may be useful in the detection of intraocular foreign bodies (Fig. 19.24), globe rupture, suprachoroidal haemorrhage and retinal detachment. It is also helpful in planning surgical repair, for example regarding placement of infusion ports during vitrectomy and whether drainage of suprachoroidal haemorrhage is required.
   d. ***Electrophysiological tests*** may be useful in assessing the integrity of the optic nerve and retina, particularly if some time has passed since the original injury and there is suspicion of a retained intraocular foreign body.

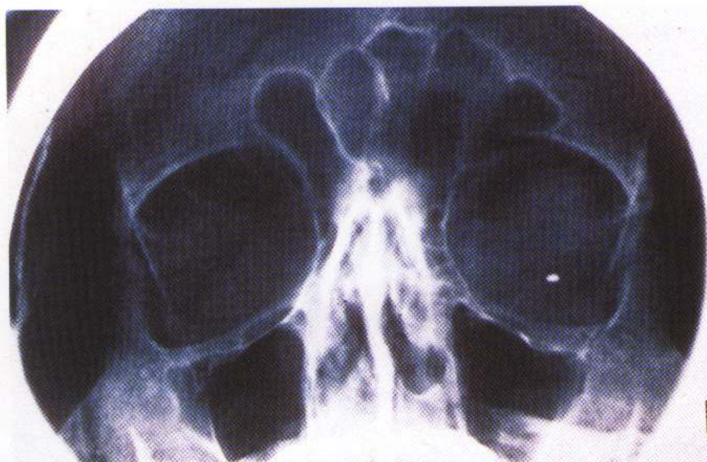


Fig. 19.22
Plain radiograph showing a left airgun pellet

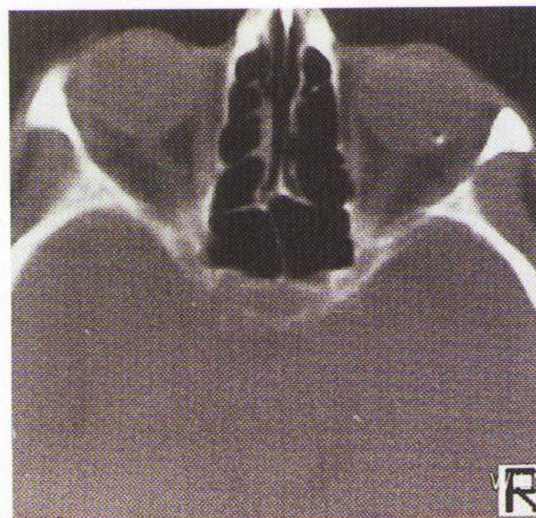


Fig. 19.23
Axial CT scan showing a left intraocular foreign body (Courtesy of Wilmer Institute)

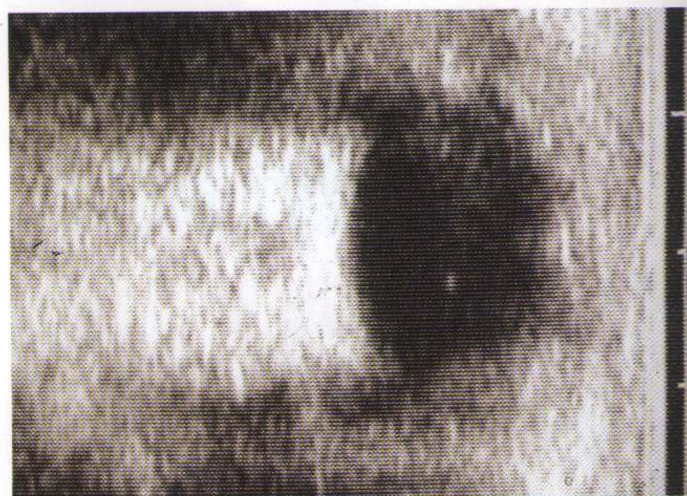
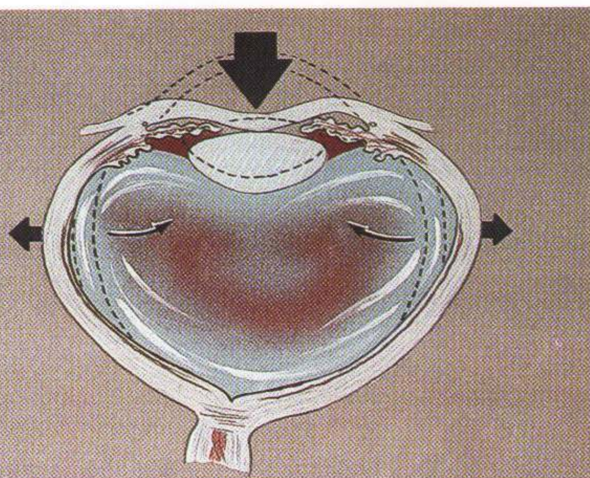


Fig. 19.24
B-scan ultrasonograph showing an intraocular foreign body

Blunt trauma

The most common causes of blunt trauma are squash balls, elastic luggage straps and champagne corks. Severe blunt trauma results in anteroposterior compression with simultaneous expansion in the equatorial plane (Fig. 19.25), associated with a transient but severe increase in intraocular pressure. Although the impact is primarily absorbed by the lens–iris diaphragm and the


Fig. 19.25
Mechanism of ocular damage by blunt trauma (Courtesy of Wilmer Institute)

vitreous base, damage can also occur at a distant site such as the posterior pole. The extent of ocular damage depends on the severity of trauma and for unknown reasons, is largely concentrated to either anterior or posterior segment. Apart from obvious ocular damage, blunt trauma commonly results in long-term effects; the prognosis is therefore necessarily guarded.

Anterior segment complications

1. **A corneal abrasion** involves a breach of the epithelium, which stains with fluorescein. If over the pupillary area, vision may be grossly impaired. This exquisitely painful condition is commonly treated with topical cycloplegia to promote comfort and antibiotic ointment. Although patching has been standard treatment in the past it has become apparent that the cornea heals faster with less pain when not patched.
2. **Acute corneal oedema** may develop, secondary to focal or diffuse dysfunction of the corneal endothelium. It is commonly associated with folds in Descemet membrane and stromal thickening, which clear spontaneously.
3. **Hyphaema** (haemorrhage into the anterior chamber) is a common complication. The source of the bleeding is the iris or ciliary body. Characteristically, the red blood cells sediment inferiorly with a resultant 'fluid' level, the height of which should be measured and documented (Fig. 19.26). Most traumatic hyphaemas are innocuous and transient and merely require daily observation until they resolve spontaneously. The immediate risk is that of secondary haemorrhage, often larger than the original hyphaema, which may occur at any time up to a week after the original injury (most commonly within the first 24 hours). The main aims of treatment are therefore prevention of secondary haemorrhage, control of any elevation of intraocular pressure and management of associated complications. Oral tranexamic acid 25 mg/kg t.i.d., an antifibrolytic agent, is useful for the former. Opinions vary, but it would appear sensible to immobilize the pupil in the dilated state, with atropine to prevent further haemorrhage. Hospital admission for a few days may be advisable so that intraocular pressure may be monitored and, if elevated, treated appropriately so as to prevent secondary corneal blood-staining (*see* Chapter 9). Traumatic uveitis is treated with topical steroids and mydriatics.
4. **The anterior uvea** may manifest structural and/or functional damage.
   a. Pupil. Severe contusion is often accompanied by transient miosis evidenced by the pattern of pigment imprinting on the anterior lens capsule (Vossius ring), which corresponds to the size of the miosed pupil (*see* Fig. 8.19). Alternatively, damage to the iris sphincter may result in traumatic mydriasis, which is often permanent: the pupil reacts sluggishly or not at all to both light and accommodation. Radial tears in the pupillary margin are common (Fig. 19.27).
   b. Iridodialysis is a dehiscence of the iris from the ciliary body at its root. The pupil is typically 'D' shaped and the dialysis is seen as a dark biconvex area near the limbus (Fig. 19.28). An iridodialysis may be asymptomatic if


Fig. 19.26
Traumatic hyphaema


Fig. 19.27
Rupture of the iris sphincter



Fig. 19.28
Inferior iridodialysis

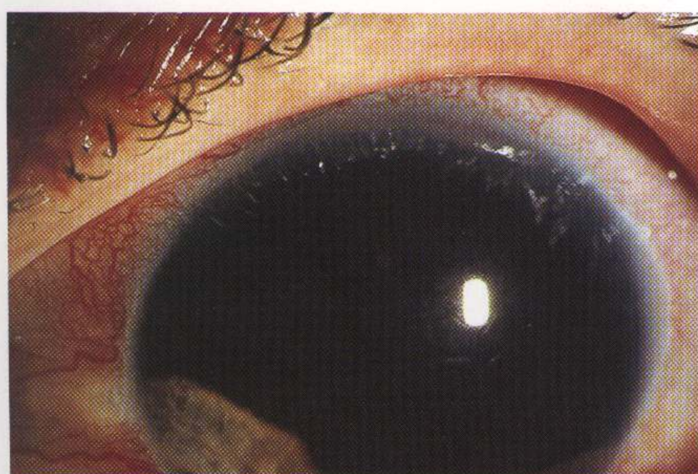


Fig. 19.29
Traumatic aniridia (Courtesy of Wilmer Institute)

covered by the upper lid; if exposed in the palpebral aperture, uniocular diplopia and glare may ensue, sometimes necessitating surgical repair of the dehiscence. Traumatic aniridia (360° iridodialysis) may rarely occur (Fig. 19.29).

c. The ciliary body may react to severe blunt trauma by temporary cessation of aqueous secretion (ciliary shock) resulting in ocular hypotony. Tears extending into the face of the ciliary body (angle recession) (Fig. 19.30) are associated with a risk of late glaucoma (*see* Chapter 9).

5. Lens

a. Cataract formation is a common sequel to blunt trauma. Postulated mechanisms include traumatic damage to the lens fibres themselves and minute ruptures in the lens capsule with influx of aqueous humour, hydration of lens fibres and consequent opacification. A ring-shaped faint anterior subcapsular opacity may underly Vossius ring. Commonly opacification occurs in the posterior subcapsular cortex along the posterior sutures ('rosette-shaped' cataract) (*see* Fig. 8.20) which may subsequently disappear, remain stationary or progress to maturity. Cataract surgery may be necessary for visually significant opacity.

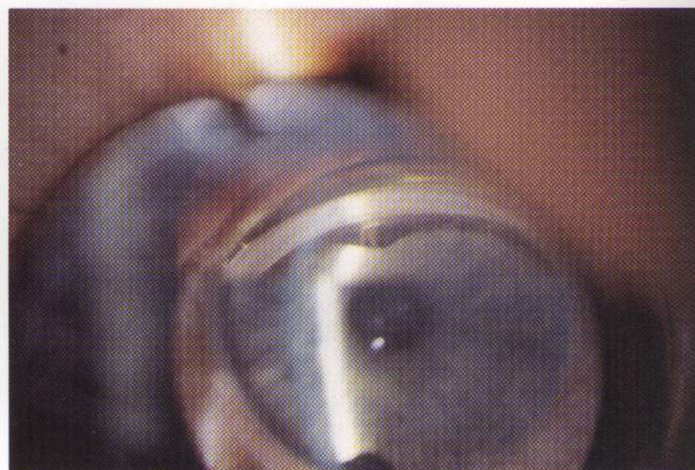


Fig. 19.30
Severe angle recession

b. Subluxation of the lens may occur, secondary to tearing of the suspensory ligament. A subluxated lens tends to deviate towards the meridian of intact zonule; the anterior chamber may deepen over the area of zonular dehiscence, if the lens rotates posteriorly. The edge of a subluxated lens may be visible under mydriasis and the iris may tremble on ocular movement (iridodonesis). Subluxation of magnitude sufficient to render the pupil partly aphakic may result in uniocular diplopia; additional lenticular astigmatism due to lens tilt may occur.

c. Dislocation due to 360° rupture of the zonule is rare and may be into the vitreous, or less commonly into the anterior chamber (Fig. 19.31).

6. Rupture of the globe may result from very severe blunt trauma. The rupture is usually anterior, in the vicinity of Schlemm canal, with prolapse of intraocular structures such as lens, iris, ciliary body and vitreous (Fig. 19.32). Occasionally, the rupture is posterior (occult) with little visible damage to the anterior segment. Clinically, occult rupture should be suspected if there is asymmetry of anterior chamber depth and intraocular pressure in the

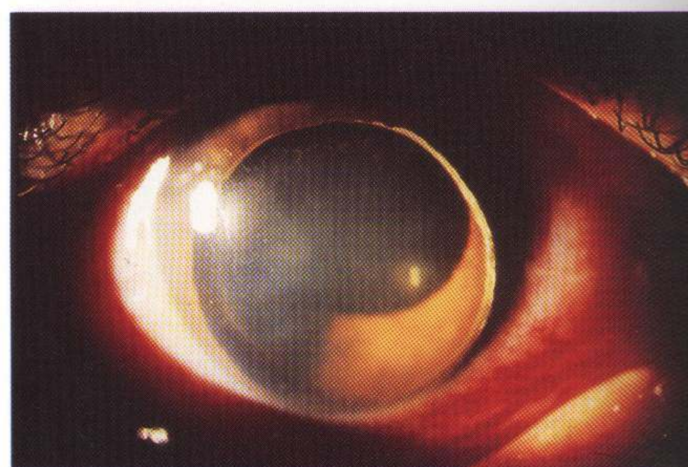


Fig. 19.31
Lens dislocation into the anterior chamber

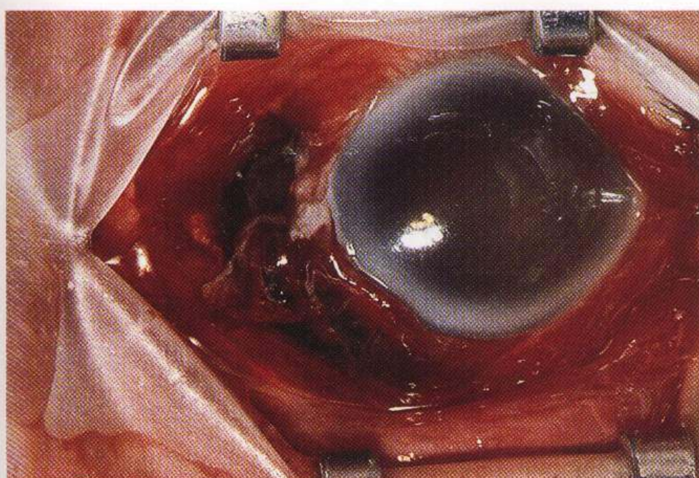


Fig. 19.32
Rupture of the globe (Courtesy of Wilmer Institute)

affected eye is low. The principles of repairing scleral ruptures are described later.

Posterior segment complications

1. **Posterior vitreous detachment**, which may be associated with vitreous haemorrhage, may be seen. Pigment cells similar to tobacco dust may be seen floating in the anterior vitreous.
2. **Commotio retinae** indicates concussion of the sensory retina resulting in cloudy swelling which gives the involved area a grey appearance. Commotio most frequently involves the temporal fundus (Fig. 19.33) and occasionally the macula, when a 'cherry red spot' may be seen at the fovea (Fig. 19.34). The prognosis in mild cases is good with spontaneous resolution without sequelae within 6 weeks. Severe involvement of the macula may be associated with intraretinal haemorrhage. Subsequent post-traumatic macular changes include progressive pigmentary degeneration and macular hole formation.
3. **Choroidal rupture** involves the choroid, Bruch membrane and RPE; it may be direct or indirect. Direct ruptures are located anteriorly at the site of impact and run parallel with the ora serrata. Indirect ruptures occur opposite the site of impact. A fresh rupture may be partially obscured by subretinal haemorrhage (Fig. 19.35), which may break through the internal limiting membrane with resultant subhyaloid or vitreous haemorrhage. Weeks to months later, on absorption of the blood, a white crescent-shaped, vertical streak of exposed underlying sclera, frequently involving the macula, concentric with the optic disc becomes visible (Fig. 19.36). The visual prognosis is poor if the fovea is involved. An uncommon late complication is secondary choroidal neovascularization which may result in haemorrhage, scarring and further visual deterioration.

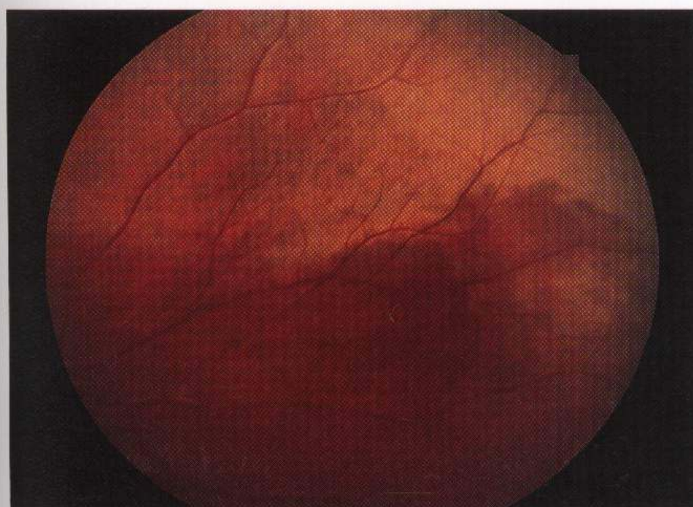


Fig. 19.33
Peripheral commotio retinae

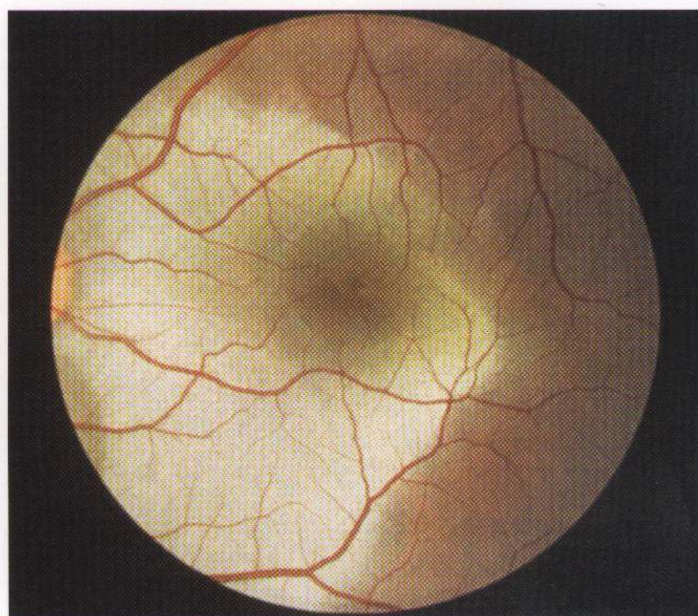


Fig. 19.34
Commotio retinae involving the macula

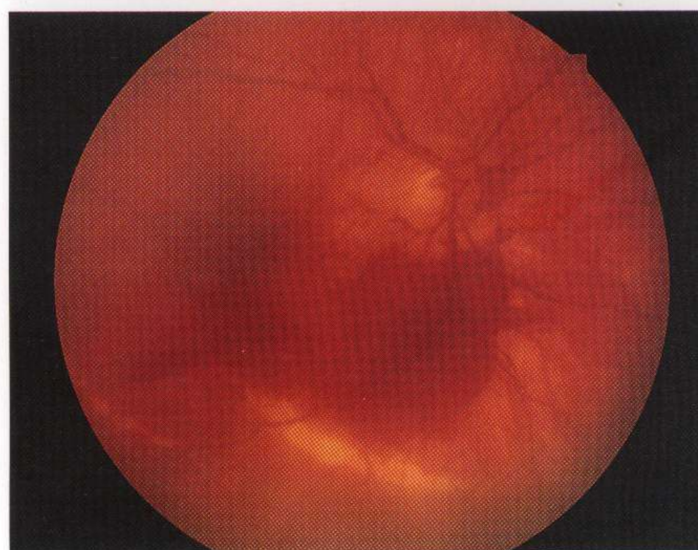


Fig. 19.35
Acute choroidal rupture with subretinal haemorrhage

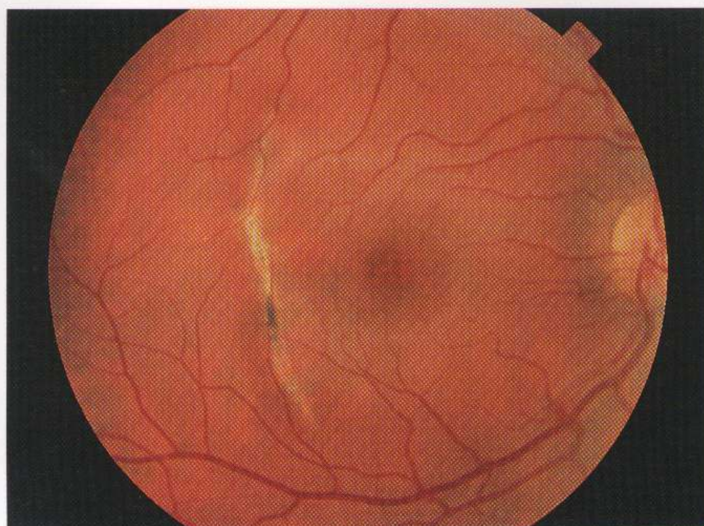


Fig. 19.36
Old choroidal rupture

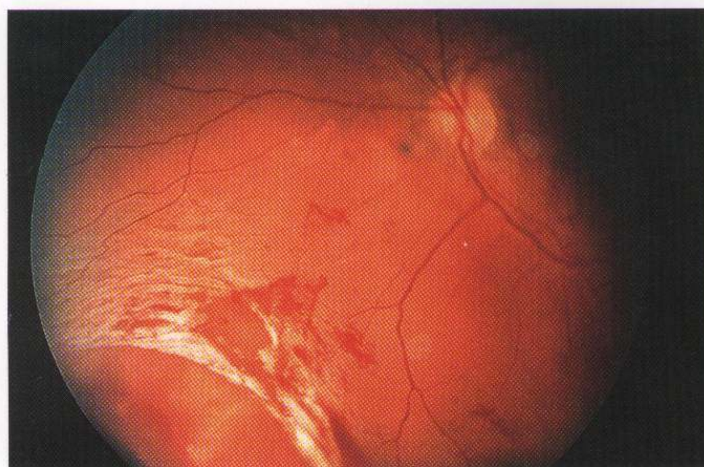


Fig. 19.37
Traumatic retinal dialysis (Courtesy of Wilmer Institute)



Fig. 19.38
Avulsion of the vitreous base

4. **Retinal breaks**, which may lead to retinal detachment, are of three main types:
   a. *Retinal dialyses* (Fig. 19.37) are caused by traction by the relatively inelastic vitreous gel along the posterior aspect of the vitreous base. The vitreous base may become avulsed, giving rise to a 'bucket-handle' appearance which comprises a strip of ciliary epithelium, ora serrata and the immediate post-oral retina into which basal vitreous gel remains inserted (Fig. 19.38). Traumatic dialyses may occur in any quadrant but are most frequent in the upper nasal, perhaps because trauma is frequently from an inferotemporal direction. Although they occur at the time of injury, any subsequent retinal detachment usually does not develop until several months later. Progression is slow, probably because the vitreous gel is healthy.
   b. *Equatorial tears* are less frequent and due to direct retinal disruption at the point of scleral impact. They may occasionally extend for more than one quadrant (giant tears).
   c. *Macular holes* may occur either at the time of injury or later following the resolution of commotio retinae.
5. **Optic nerve**
   a. *Optic neuropathy* is an uncommon but often devastating cause of permanent visual loss following contusive injuries to the head, particularly the forehead. This impact is thought to transmit a shock wave to the optic canal, damaging the optic nerve. Typically the optic nerve head and fundus are initially normal, the only objective finding being a relative afferent pupillary defect. Neither systemic steroids nor surgical decompression of the optic canal prevent the development of optic atrophy within 3–4 weeks.
   b. *Optic nerve avulsion* is rare and typically occurs when an object intrudes between the globe and the orbital wall, displacing the eye. Postulated mechanisms include sudden extreme rotation or anterior displacement of the globe. Avulsion may be isolated or occur in

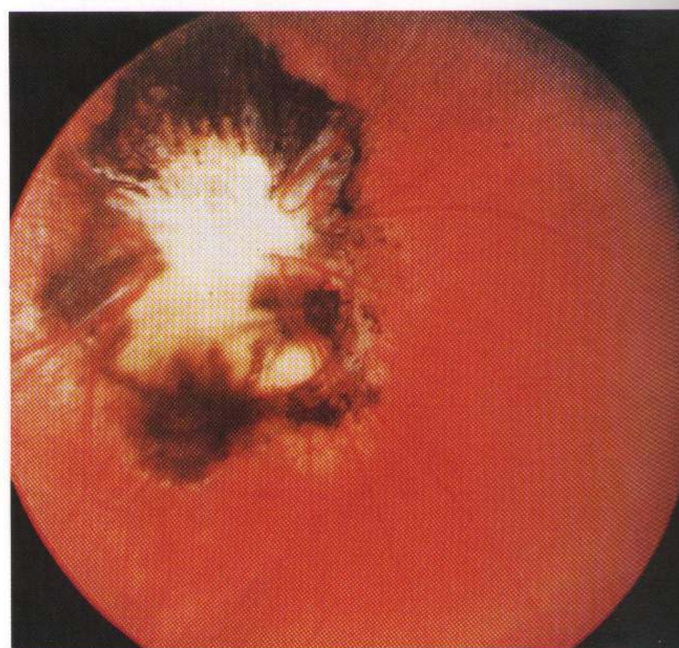


Fig. 19.39
Avulsed optic nerve (Courtesy of E.M. Eagling, M.J. Roper-Hall, Eye Injuries, Butterworths, 1986)

association with other injuries to the globe or orbit. Fundus examination shows a striking cavity where the optic nerve head has retracted from its dural sheath (Fig. 19.39). There is no treatment; the visual prognosis depends on whether avulsion is partial or complete.

Non-accidental injury

Non-accidental injury (shaken baby syndrome) indicates physical abuse in children usually under the age of 2 years, and should be suspected whenever characteristic ophthalmic features are identified in the absence of a convincing alternative explanation. The diagnosis should be considered with the help of a specialist paediatrician; most hospitals dealing with children will have a child abuse team. The injury may be caused by violent shaking alone. However, careful examination also frequently reveals signs of impact injuries. Brain damage is thought to be the result of hypoxia and ischaemia resulting from apnoea, rather than shearing or impact.

1. **Presentation** is frequently with irritability, lethargy and vomiting, which may be initially misdiagnosed as gastroenteritis or other infection because the history of injury is withheld.
2. **Systemic features** include subdural haematoma and impact injuries to the head, ranging from skull fractures to soft tissue bruises. Many survivors suffer significant permanent neurological handicap.
3. **Ocular features** are many and varied. The most important are as follows:
   - Retinal haemorrhages, unilateral or bilateral, are the most common feature. The haemorrhages typically involve different layers of the retina and are most obvious in the posterior pole (Fig. 19.40), although they often extend to the periphery.
   - Periocular bruising and subconjunctival haemorrhages.
   - Poor visual responses and afferent pupillary defects.
   - Visual loss occurs in about 20% of cases largely as a result of cerebral damage.

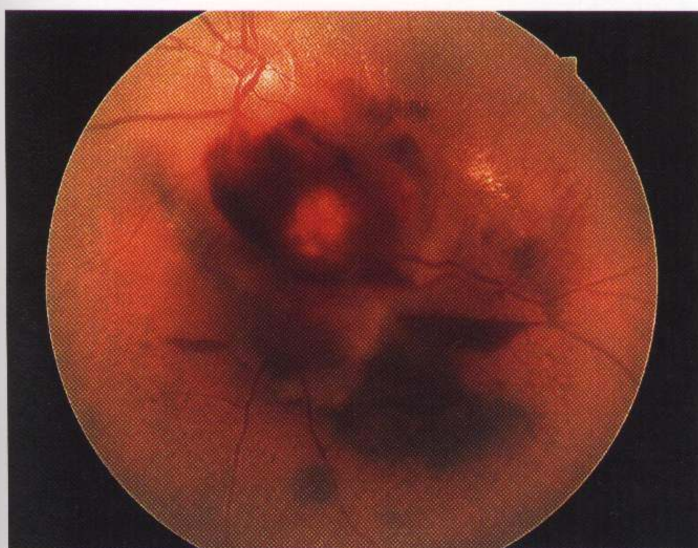


Fig. 19.40
Haemorrhages in non-accidental injury

Penetrating trauma

Causes

Penetrating injuries are three times more common in males than females, and in the younger age group. The most frequent causes are assault, domestic accidents and sport. The extent of the injury is determined by the size of the object, its speed at the time of impact and its composition. Sharp objects such as knives cause well-defined lacerations of the globe. However, the extent of damage caused by flying foreign bodies is determined by their kinetic energy. For example, an airgun pellet is large and, although relatively slow moving, has a high kinetic energy and can thus cause considerable ocular damage. In contrast, a fast-moving fragment of shrapnel has a low mass and therefore will cause a well-defined laceration with relatively less intraocular damage than an airgun pellet.

> *NB:* Of paramount immediate importance is the introduction of infection with any penetrating injury. Endophthalmitis or panophthalmitis, often more severe than the initial injury, may ensue with loss of the eye.

Tractional retinal detachment

Tractional retinal detachment may occur secondary to vitreous incarceration in the wound and intragel vitreous haemorrhage (Fig. 19.41a), which stimulates fibroblastic proliferation along the planes of incarcerated vitreous (Fig. 19.42). Subsequent contraction of such membranes leads to shortening and a rolling effect on the peripheral retina in the region of the vitreous base and eventually to tractional retinal detachment (*see* Fig. 19.41b).

Principles of primary repair

The technique of primary repair depends on the severity of the wound and any associated complications such as iris

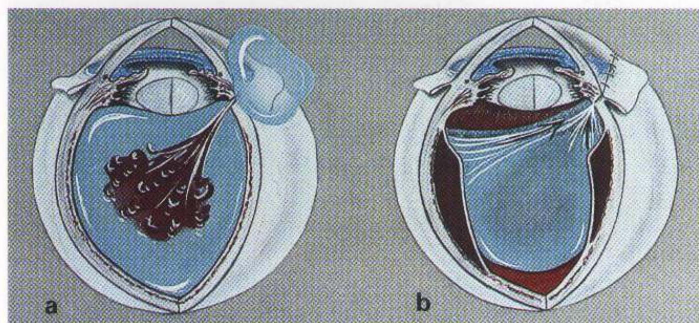


Fig. 19.41
(a) Penetrating injury resulting in vitreous prolapse and intraocular haemorrhage; **(b)** subsequent proliferation and vitreoretinal traction resulting in retinal detachment (Courtesy of Wilmer Institute)

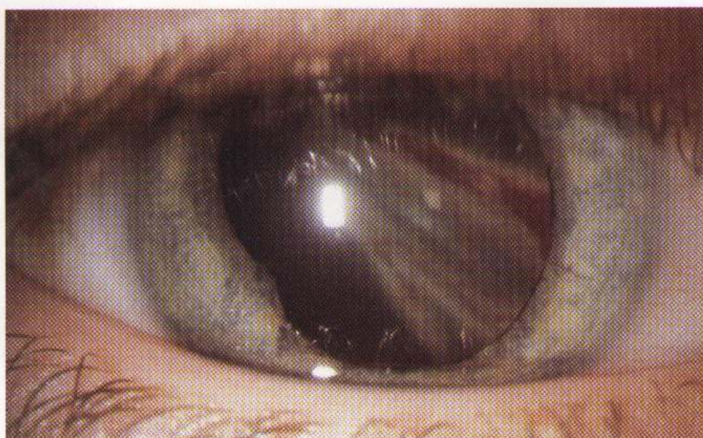

Fig. 19.42
Retrolental fibrous proliferation following penetrating trauma


Fig. 19.45
Penetrating corneal wound with a flat anterior chamber


Fig. 19.43
Penetrating corneal wound with a formed anterior chamber (Courtesy of Wilmer Institute)

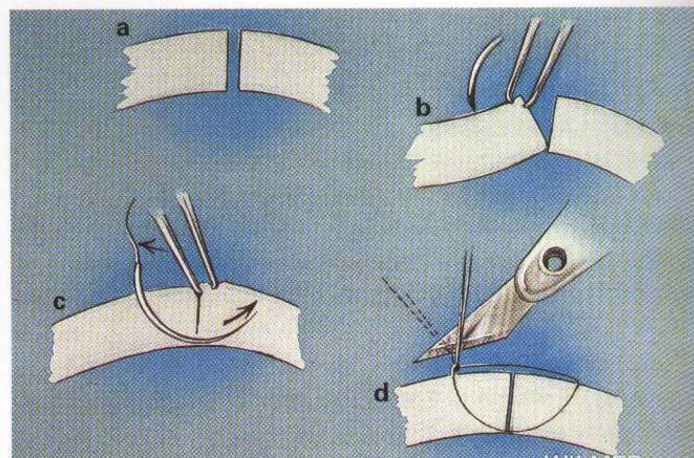


Fig. 19.46
Technique of repair of a penetrating corneal wound. The anterior chamber may be deepened with a viscoelastic substance (Courtesy of Wilmer Institute)

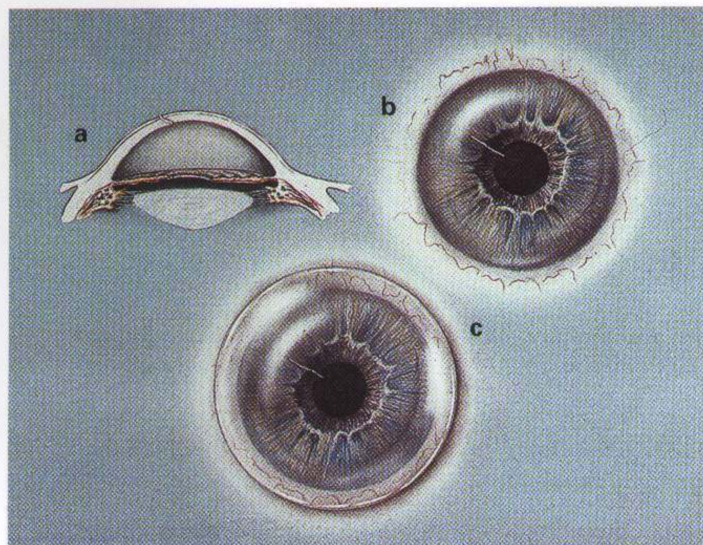


Fig. 19.44
(a and b) Penetrating corneal wound with a formed anterior chamber; **(c)** bandage contact lens in situ (Courtesy of Wilmer Institute)

incarceration, flat anterior chamber and damage to intraocular contents.

1. **Small shelving corneal lacerations** (Figs 19.43 and 19.44a and b) with formed anterior chamber may not require suturing as they often heal spontaneously or with the aid of a soft bandage contact lens (Fig. 19.44c).
2. **Medium-sized corneal lacerations** usually require suturing, especially if the anterior chamber is shallow or flat (Fig. 19.45). The technique of suturing is shown in Figs 19.46 and 19.47. If the laceration involves the limbus it is important to expose the adjacent sclera (Fig. 19.48) and to suture any scleral extension (Fig. 19.49). A shallow anterior chamber may re-form spontaneously once the cornea has been sutured, if not it should be reformed with balanced salt solution. A postoperative bandage contact lens may also be useful for a few days to ensure that the anterior chamber remains deep.
3. **Corneal lacerations with iris involvement** (Fig. 19.50 and 19.51a). Management depends on the duration and extent of incarceration.

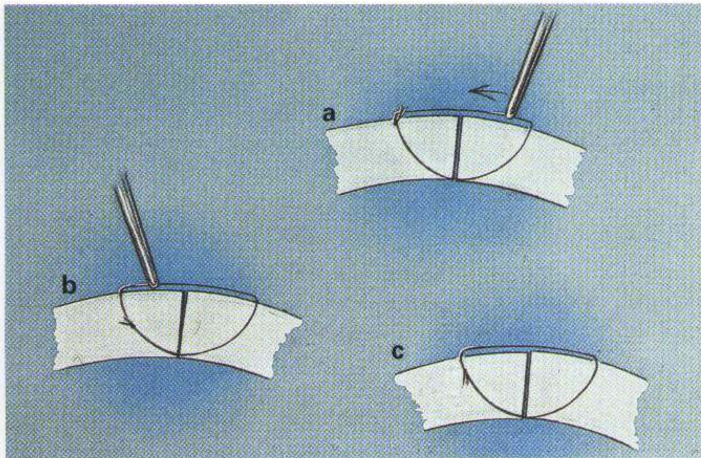


Fig. 19.47
Technique of burying the knot (Courtesy of Wilmer Institute)



Fig. 19.50
Penetrating corneal wound with iris prolapse (Courtesy of Wilmer Institute)

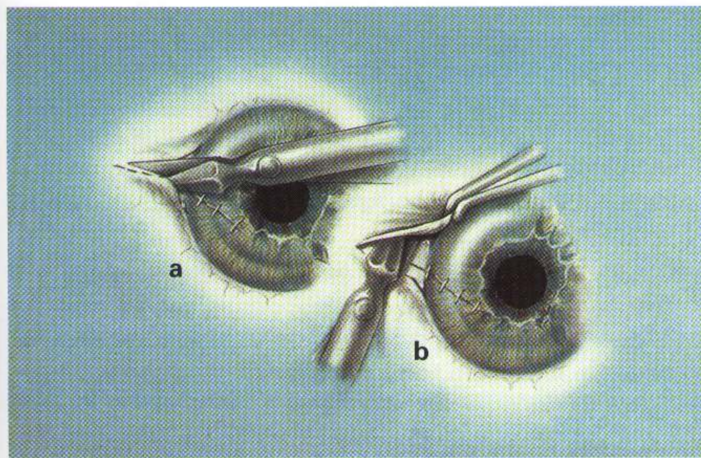


Fig. 19.48
Exploration of the sclera following repair of a corneal laceration (Courtesy of Wilmer Institute)

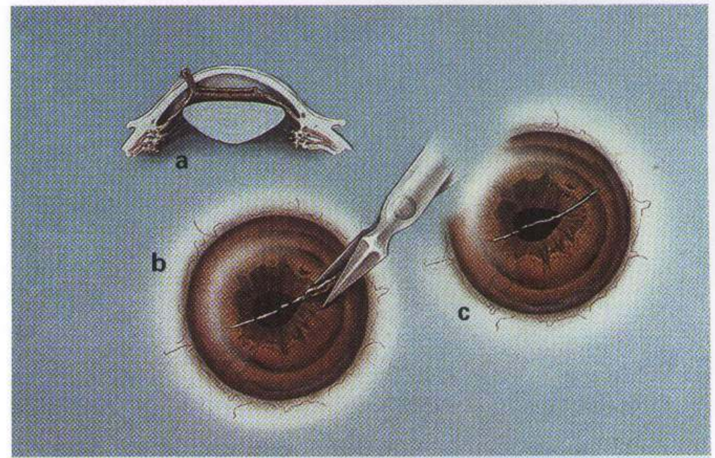


Fig. 19.51
(a) Penetrating corneal wound with iris prolapse; **(b)** abscision of prolapsed iris; **(c)** final appearance

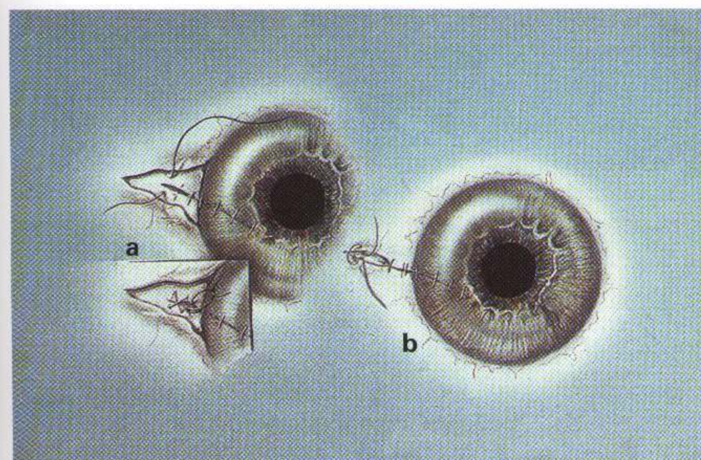


Fig. 19.49
Repair of a scleral laceration (Courtesy of Wilmer Institute)



Fig. 19.52
Corneal laceration associated with cataract

- A small peak of recently incarcerated iris may be reposited and the pupil constricted with intracameral acetylcholine.
- Large incarcerations of iris should be abscised (Fig. 19.51b), especially if of more than a few days' duration or the iris appears non-viable, due to the risk of endophthalmitis.

4. **Corneal laceration with lenticular damage** (Figs 19.52 and 19.53a) is treated by suturing the laceration and

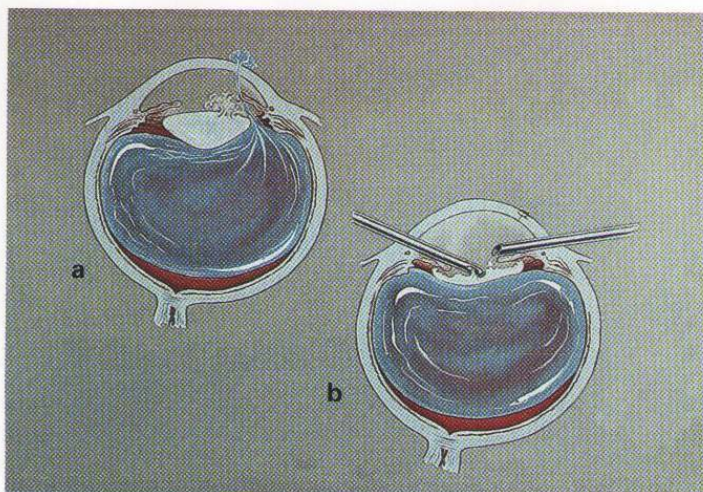


Fig. 19.53
(**a**) Penetrating wound with disruption of the lens and anterior herniation of vitreous through a ruptured zonule; (**b**) repair of corneal wound followed by lensectomy and anterior vitrectomy (Courtesy of Wilmer Institute)

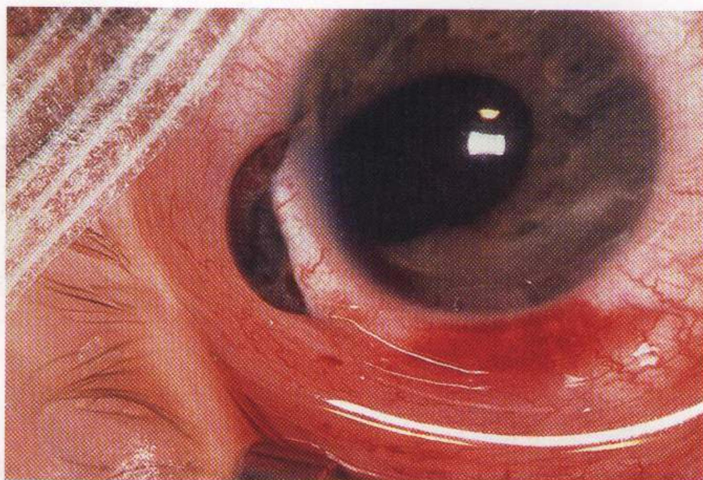


Fig. 19.54
Scleral laceration with iridociliary prolapse (Courtesy of Wilmer Institute)

removing the lens by phacoemulsification or with a vitreous cutter (Fig. 19.53b). The latter is preferred if there is associated vitreous involvement. Primary implantation of an intraocular lens is frequently associated with a favourable visual outcome and a low rate of postoperative complications.

5. **Anterior scleral lacerations** limited to the insertions of the recti (i.e. anterior to the spiral of Tillaux and therefore the ora serrata) have a better prognosis than more posterior wounds. An anterior scleral wound may, nevertheless, be associated with serious complications such as iridociliary prolapse and vitreous incarceration (Fig. 19.54). The latter, unless appropriately managed, may result in subsequent vitreoretinal traction and retinal detachment. Every attempt should be made to reposit exposed viable uveal tissue and cut prolapsed vitreous flush with the wound.

> ***NB:*** Cellulose sponges should not be used to remove vitreous for fear of inducing vitreous traction.

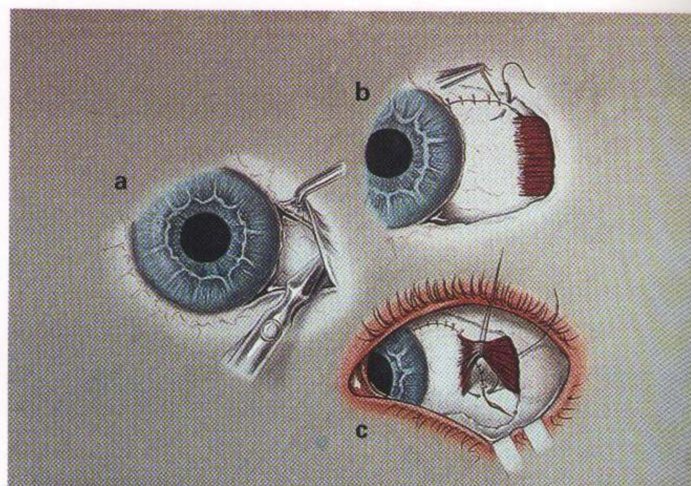


Fig. 19.55
(**a**) Exposure of sclera; (**b** and **c**) repair of scleral laceration (Courtesy of Wilmer Institute)

6. **Posterior scleral lacerations** are frequently associated with retinal breaks unless very superficial. The scleral wound is exposed (Fig. 19.55a) and sutured starting anteriorly and working posteriorly (Fig. 19.55b and c). It may also be necessary to treat retinal breaks prophylactically.

> ***NB:*** During repair it is very important not to exert excessive pressure and traction on the eye to prevent or minimize loss of intraocular contents.

Aims of secondary repair

Secondary repair of posterior segment trauma, if appropriate, is usually carried out 10–14 days after primary repair. This allows time not only for healing of wounds but also for the development of posterior vitreous separation which facilitates good vitrectomy. The main aims of secondary repair are:

- To clear opacities of the media such as cataract and vitreous haemorrhage in order to improve vision.
- To stabilize abnormal vitreoretinal interactions and thereby prevent long-term sequelae such as tractional retinal detachment.

Extraocular foreign bodies

Small foreign bodies such as particles of steel, coal or sand often impact on the corneal or conjunctival surface. Subsequent to such impact, such foreign bodies may:

- Be washed along the tear film into the lacrimal drainage system.
- Adhere to the superior palpebral conjunctiva in the subtarsal sulcus and abrade the cornea with every blink. A pathognomonic pattern of linear corneal abrasions may be seen (Fig. 19.56). A subtarsal foreign body is easily missed unless the upper lid is everted.

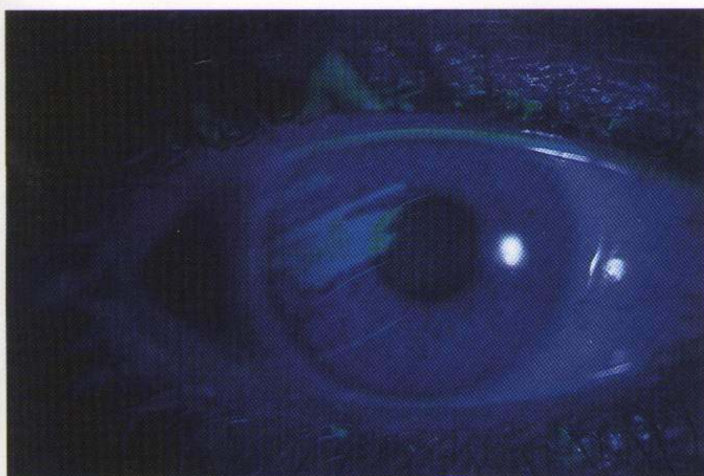


Fig. 19.56
Linear corneal abrasions stained with fluorescein due to a subtarsal foreign body

- Ascend and lodge in the superior conjunctival fornix and produce chronic conjunctivitis. Such a foreign body is easily missed, unless the lid is double-everted and the fornix is examined.
- Impact in the bulbar conjunctiva.
- Impact in the corneal epithelium or stroma to a depth proportional to the velocity of the foreign body.
- A very high-velocity foreign body may penetrate the cornea or sclera and lodge intraocularly.

Corneal foreign bodies

1. **Clinical features**. Corneal foreign bodies are extremely common and cause considerable irritation. Leukocytic infiltration may also develop around any foreign body of some duration. If a foreign body is allowed to remain, there is a risk of secondary infection and corneal ulceration. Mild secondary uveitis is common with irritative miosis and photophobia. Ferrous foreign bodies of even a few days' duration often result in rust staining of the bed of the abrasion.
2. **Management**
   a. Careful slit-lamp examination is essential to locate the exact position and depth of the foreign body.
   b. The foreign body is removed under slit-lamp visualization using a sterile 26-gauge needle. Magnetic removal may be useful for a deeply embedded metallic foreign body. A residual 'rust ring', is easiest to remove with a sterile 'burr'.
   c. Antibiotic ointment is instilled together with a cycloplegic and/or ketorolac to promote comfort.

> *NB:* Any discharge, infiltrate or significant uveitis should raise suspicion of secondary bacterial infection; subsequent management should be as for a corneal ulcer. Metallic foreign bodies are often sterile due to acute rise in temperature during transit through the air; organic and stone foreign bodies however, carry a higher risk of infection.

Intraocular foreign bodies

An intraocular foreign body may traumatize the eye mechanically, introduce infection or exert other toxic effects on the intraocular structures. Once in the eye, the foreign body may lodge in any of the structures it encounters; thus it may be located anywhere from the anterior chamber to the retina and choroid. Notable mechanical effects include cataract formation secondary to capsular injury, vitreous liquefaction, and retinal haemorrhages and tears. Stone and organic foreign bodies are particularly prone to result in infection. Many substances including glass, many plastics, gold and silver are inert. However, iron and copper may undergo dissociation and result in siderosis and chalcosis respectively.

Siderosis

Perhaps the commonest foreign body is a piece of steel. An intraocular ferrous foreign body undergoes dissociation resulting in the deposition of iron in the intraocular epithelial structures, notably the lens epithelium and the retina, where it exerts a toxic effect on cellular enzyme systems, with resultant cell death. Features of siderosis include anterior capsular cataract, consisting of radial iron deposits on the anterior lens capsule, reddish-brown staining of the iris (Fig. 19.57), secondary glaucoma due to trabecular damage and pigmentary retinopathy, the last of which has the most profound effects on vision. Electroretinography manifests progressive attenuation of the b-wave over time.

Chalcosis

The ocular reaction to an intraocular foreign body with a high copper content involves a violent endophthalmitis-like picture, often with progression to phthisis bulbi. On the other hand, an alloy such as brass or bronze, with a relatively low copper content, results in chalcosis. Electrolytically dissociated copper becomes deposited intraocularly, resulting in a picture similar to that seen in Wilson disease. Thus a

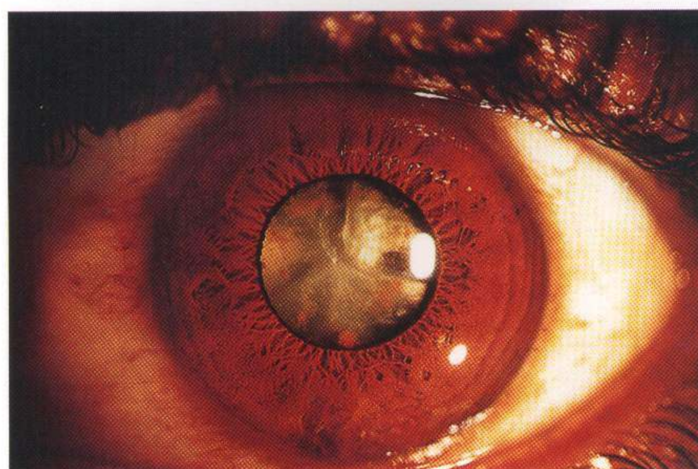


Fig. 19.57
Cataract and iris staining due to siderosis (Courtesy of J. Salmon)

Kayser–Fleischer ring develops (*see* Fig. 5.140), as does an anterior 'sunflower' cataract. Retinal deposition results in golden plaques visible ophthalmoscopically. Since copper is less retinotoxic than iron, degenerative retinopathy does not develop and visual function may be preserved.

Initial management

1. **Accurate history** is vital to determine the origin of the foreign body; it may be helpful for the patient to bring any causative objects such as a chisel.
2. **Ophthalmic examination** is performed, paying special attention to any possible sites of entry or exit. Topical fluorescein may be helpful to identify an entry wound. Alignment and projection of identified wounds may allow logical deduction of the probable location of a foreign body. Gonioscopy and funduscopy must be performed. Associated signs such as lid laceration and damage to anterior segment structures must be noted.
3. **CT** with axial and coronal cuts is used to detect and localize metallic intraocular foreign bodies (*see* Fig. 19.23), providing cross-sectional images with a sensitivity and specificity that are superior to plain radiography and ultrasonography.

> ***NB:*** MRI scanning is contraindicated in the context of a metallic intraocular foreign body.

Technique of removal

1. **Magnet** removal is as follows:
   a. A sclerotomy is performed adjacent to the foreign body (Fig. 19.58a).
   b. Low-intensity diathermy is applied to the choroid to prevent bleeding (Fig. 19.58b).
   c. The foreign body is removed with the magnet (Fig. 19.58c).
   d. Cryotherapy may be applied to the induced retinal tear and adjacent retina (Fig. 19.58d).
   e. Scleral buckling (Fig. 19.58e) may be performed to reduce the risk of retinal detachment, but this is optional.
2. **Forceps** removal may be used for non-magnetic foreign bodies and magnetic foreign bodies that cannot be safely removed with a magnet.
   a. A complete pars plana vitrectomy is performed (Fig. 19.59a).
   b. Intraocular forceps are introduced (Fig. 19.59b).
   c. A small foreign body may be removed through the pars plana.
   d. A large foreign body in the aphakic eye may be removed by holding it in the pupillary space (Fig. 19.60a), inserting a keratome (Fig. 19.60b) and delivering it through a limbal incision (Fig. 19.60c).

> ***NB:*** Prophylaxis of endophthalmitis with intravitreal antibiotics is required in high-risk cases such as soil-contaminated or vegetable matter foreign bodies.

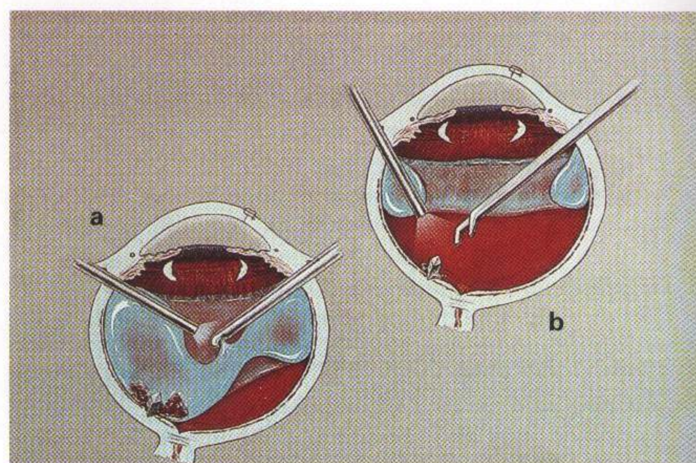


Fig. 19.59
Forceps removal of intraocular foreign body through the pars plana (*see text*) (Courtesy of Wilmer Institute)

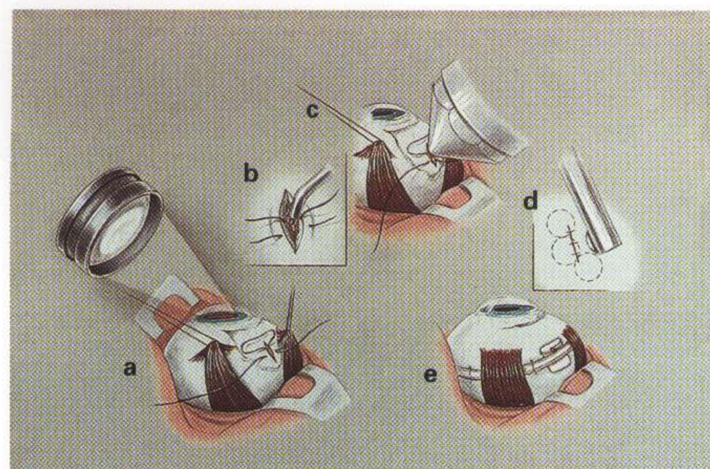


Fig. 19.58
Magnet removal of intraocular foreign body (*see text*) (Courtesy of Wilmer Institute)

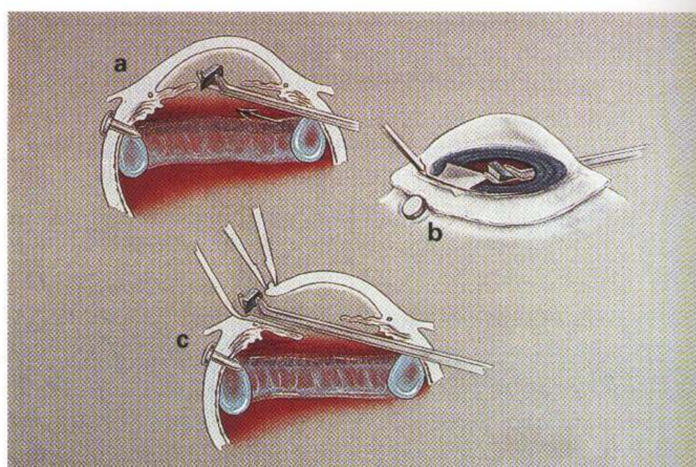


Fig. 19.60
Forceps removal of intraocular foreign body through the limbus (*see text*) (Courtesy of Wilmer Institute)

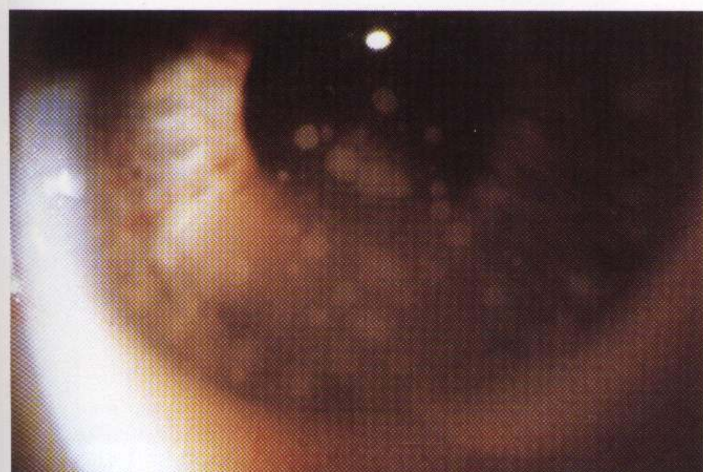

Fig. 19.61
Extremely damaged globe where primary enucleation is justifiable

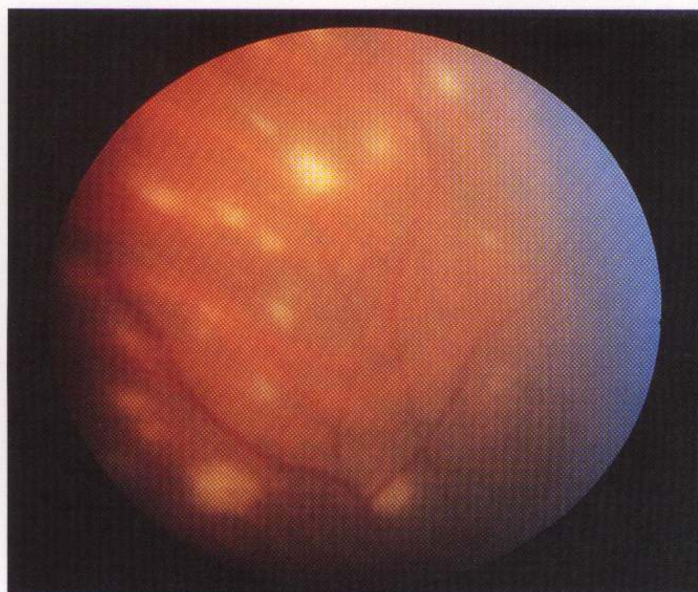
Enucleation

Primary enucleation should be performed only for very severe injuries, with no prospect of retention of vision when it is impossible to repair the sclera (Fig. 19.61). Secondary enucleation may be considered following primary repair if the eye is severely and irreversibly damaged, particularly if it is also unsightly and uncomfortable. The time delay also allows the patient valuable time to mentally and emotionally adapt to the prospect of losing an eye. Based on anecdotal evidence, it has been recommended that enucleation should be performed within 10 days of the original injury in order to prevent the very remote possibility of sympathetic ophthalmitis. However, objective evidence for this is lacking.

Sympathetic ophthalmitis

Sympathetic ophthalmitis is a very rare, *bilateral*, granulomatous panuveitis which occurs after penetrating ocular trauma usually associated with uveal prolapse or, less frequently, following intraocular surgery. The traumatized eye is referred to as the *exciting* eye and the fellow eye, which also develops uveitis, is called the *sympathizing* eye.


Fig. 19.62
Mutton fat keratic precipitates in sympathetic ophthalmitis


Fig. 19.63
Multifocal choroiditis in sympathetic ophthalmitis (Courtesy of J. Salmon)

1. **Presentation** in 65% of cases is between 2 weeks and 3 months after initial injury; 90% of all cases occur within the first year.
2. **Signs**
   - The exciting eye shows evidence of the initial trauma and is frequently very red and irritable.
   - The sympathizing eye becomes photophobic and irritable.
   - Both eyes then develop a chronic granulomatous anterior uveitis with iris nodules and mutton fat keratic precipitates (Fig. 19.62).
   - Optic disc swelling and multifocal choroiditis involve the entire fundus (Fig. 19.63).
3. **Course**. Rarely the uveitis runs a relatively mild and self-limiting course. Usually, however, the intraocular inflammation becomes chronic and, unless appropriately treated, may lead to cataract, glaucoma and phthisis bulbi.
4. **Treatment** with systemic steroids is usually effective and the long-term visual prognosis good. A variety of steroid-sparing immunosuppressive agents are also beneficial.

Chemical injuries

Introduction

Causes

Chemical injuries range in severity from trivial to potentially blinding. The majority are accidental and a few the result of assault. Two-thirds of accidental burns occur at work and the remainder at home. Alkali burns are twice as common as

acid burns since alkalis are more widely used at home and in industry. The most common involved alkalis are ammonia, sodium hydroxide and lime. The commonest acids implicated are sulphuric, sulphurous, hydrofluoric, acetic, chromic and hydrochloric. The severity of a chemical injury is related to the properties of the chemical, the area of affected ocular surface, duration of exposure (retention of particulate chemical on the surface of the globe) and related effects such as thermal damage. Alkalis tend to penetrate deeper than do acids, which coagulate surface proteins, resulting in a protective barrier. Ammonia and sodium hydroxide may produce severe damage because of rapid penetration. Hydrofluoric aid used in glass etching and cleaning also tends to rapidly penetrate the eye, while sulphuric acid may be complicated by thermal effects and high-velocity impact after car battery explosions.

Pathophysiology

1. **Ocular damage** by severe chemical injuries occurs in the following order:
   - Necrosis of the conjunctival and corneal epithelium with disruption and occlusion of the limbal vasculature. Loss of limbal stem cells may subsequently result in conjunctivalization and vascularization of the corneal surface or persistent corneal epithelial defects with sterile corneal ulceration and perforation. Other long-term effects include ocular surface wetting disorders, symblepharon formation and cicatricial entropion.
   - Deeper penetration causes breakdown and precipitation of glycosaminoglycans and stromal corneal opacification.
   - Anterior chamber penetration results in iris and lens damage.
   - Ciliary epithelial damage impairs secretion of ascorbate, which is required for collagen production and corneal repair.
   - Hypotony and phthisis bulbi may ensue.
2. **Healing** of the corneal epithelium and stroma is as follows:
   - The epithelium heals by migration of epithelial cells which originate from limbal stem cells.
   - Damaged stromal collagen is phagocytosed by keratocytes and new collagen is synthesized.

Management

Emergency treatment

A chemical burn is the only eye injury that requires immediate treatment without first taking a history and performing a careful examination. Immediate treatment is as follows:

1. **Copious irrigation** is crucial to minimize duration of contact with the chemical and normalize the pH in the conjunctival sac as soon as possible. Normal saline (or equivalent) should be used to irrigate the eye for 15–30 minutes or until pH is normalized.
2. **Double-eversion of the eyelids** should be performed so that any retained particulate matter trapped in the fornices, such as lime or cement, may be removed.
3. **Debridement** of necrotic areas of corneal epithelium should be performed to allow for proper re-epithelialization.

Grading of severity

Acute chemical injuries are then graded to plan appropriate subsequent treatment and afford an indication of likely ultimate prognosis. Grading is performed on the basis of corneal clarity and severity of limbal ischaemia. The latter is assessed by observing the patency of the deep and superficial vessels at the limbus (Fig. 19.64).

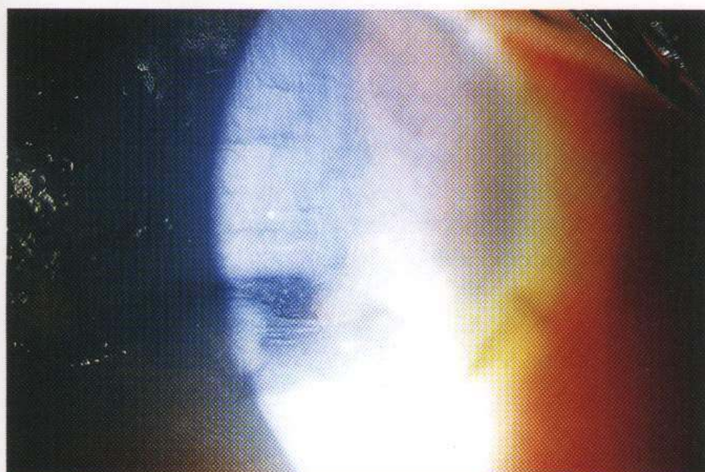
1. **Grade I**: clear cornea and no limbal ischaemia (excellent prognosis).
2. **Grade II**: hazy cornea but with visible iris details (Fig. 19.65) and less than one-third (120°) of limbal ischaemia (good prognosis).

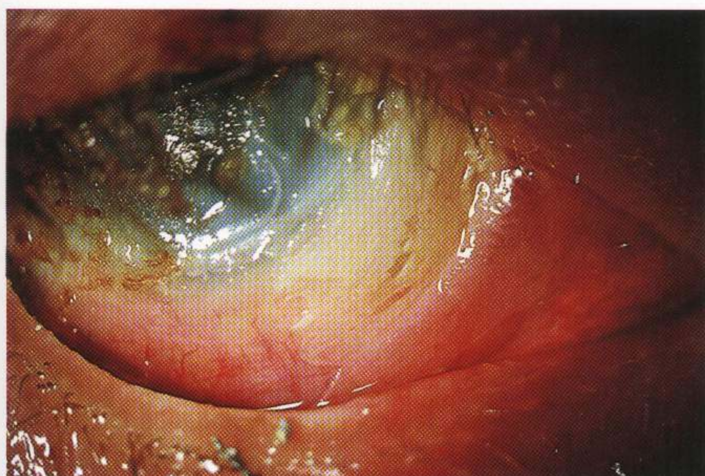


Fig. 19.64
Severe limbal ischaemia following a recent chemical injury



Fig. 19.65
Grade II chemical injury with corneal haze but visible iris details


Fig. 19.66
Grade III chemical injury with corneal haze obscuring iris details


Fig. 19.67
Grade IV chemical injury with totally opaque cornea

3. **Grade III**: total loss of corneal epithelium, stromal haze obscuring iris details (Fig. 19.66) and between one-third and half (120° to 180°) of limbal ischaemia (guarded prognosis).
4. **Grade IV**: opaque cornea and more than half (>180°) of limbal ischaemia (Fig. 19.67) (very poor prognosis).

Other features to note at initial assessment are the extent of corneal and conjunctival epithelial loss, iris changes, status of the lens and intraocular pressure.

Medical treatment

Mild (grades I and II) injuries are treated with a short course of topical steroids, cycloplegics and prophylactic antibiotics for about 7 days. The main aims of treatment of more severe burns are to reduce inflammation, promote epithelial regeneration and prevent corneal ulceration.

1. **Steroids** reduce inflammation and neutrophil infiltration. However, they also impair stromal healing by reducing collagen synthesis and inhibiting fibroblast migration. For this reason topical steroids may be used initially but must be tailed off after 7–10 days when sterile corneal ulceration is most likely to occur. They may be replaced by topical NSAIDs, which do not affect keratocyte function.
2. **Ascorbic acid** reverses a localized tissue scorbutic state and improves wound healing by promoting the synthesis of mature collagen by corneal fibroblasts. Topical sodium ascorbate 10% is given 2-hourly in addition to a systemic dose of 2 g q.i.d.
3. **Citric acid** is a powerful inhibitor of neutrophil activity and reduces the intensity of the inflammatory response. Chelation of extracellular calcium by citrate also appears to inhibit collagenase. Topical sodium citrate 10% is given 2-hourly for about 10 days. The aim is to eliminate the second wave of phagocytes, which normally occurs 7 days after the injury.
4. **Tetracyclines** are collagenase inhibitors and also inhibit neutrophil activity and reduce ulceration. They are administered both topically and systemically (e.g. doxycycline 100 mg b.d.).

Surgery

1. **Early surgery** may be necessary to revascularize the limbus, restore the limbal cell population and re-establish the fornices. One or more of the following procedures may be used:
   - Advancement of Tenon capsule and suturing to the limbus is aimed at re-establishing limbal vascularity, thus preventing the development of corneal ulceration.
   - Limbal stem cell transplantation from the patient's other eye (autograft) or from a donor (allograft) is aimed at restoring normal corneal epithelium.
   - Amniotic membrane grafting to promote epithelialization and suppression of fibrosis.
2. **Late surgery** may involve the following procedures:
   - Division of conjunctival bands (Fig. 19.68) and symblepharon (Fig. 19.69).

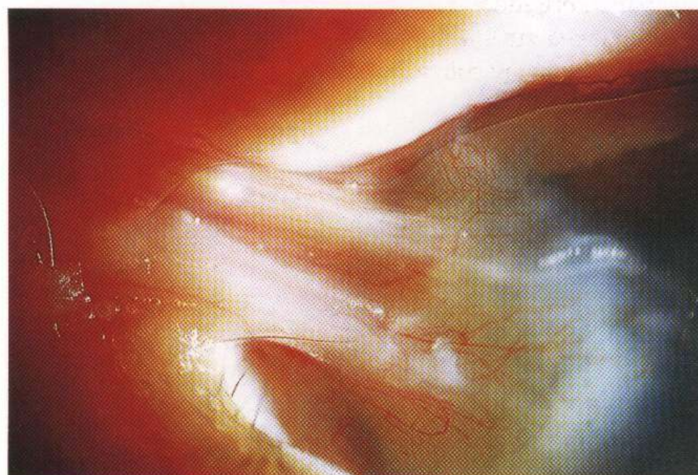

Fig. 19.68
Conjunctival adhesions following chemical injury



Fig. 19.69
Symblepharon following chemical injury

- Conjunctival or mucous membrane grafts.
- Correction of eyelid deformities.
- Keratoplasty should be delayed for at least 6 months and preferably longer to allow maximal resolution of inflammation.
- Keratoprostheses (*see* Fig. 6.15) may be required in very severely damaged eyes because the results of conventional grafting are poor.